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Editorial

Ebola virus Epidemiology in West Africa

Maria Teresa Mascellino*[1], De Angelis M[1], Borgese L[1]

[1]*Sapienza University of Rome, Italy*

**Corresponding author: Dr. Maria Teresa Mascellino, Sapienza University of Rome (Italy), Piazzale Aldo Moro 5, 00185 ROME (Italy), Tel: +39-06-49970880; Fax: +39-06-49972628; Email: mariateresa.mascellino@uniroma1.it*

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The outbreak of Ebola virus (EBOV) first identified in the forested southeast in Guinea in mid-March 2014 and then spread to the capital Konakry, was first described by Chandler C. [1] who affirmed that up to April of the same year more than 80 people have died.

Following these early observations, the scientists tried to understand the mechanism of the onset of emerging infectious diseases in general and specifically of the present zoonosis that was devastating wide regions of West Africa.

The first official case of epidemic in West Africa has been notified to WHO (World Health Organization) in March 2014. It involved a two years old child, Emile Ouamouno , dead in December 2013 in Meliandou village in the prefecture of Guinea close to the forest between Sierra Leone and Liberia. Afterwards, the infection would have been transmitted to a physician and to his family and then the focus would have moved towards bigger cities up to the capital Konakri [2].

The EBOV epidemic as well as other diseases that occur in Africa are correlated to the poverty. In fact with the exception of the physician, the spread of the disease occurred in regions at a very low social-economic standard.

Probably this apparently sudden human infection by Ebola was present in West Africa even before the cases reported above but due to the high rate of mortality detected in this population, they were omitted and not correctly diagnosed. In fact some AA [3] have reported that the most part of people in rural population of Gabon showed a high prevalence of both humoral and cellular immunity to Zaire Ebola virus. Similar results were obtained even by Gonzales et al. in 1989 [4] in randomized representative central african population. Some anthropologists also reported few cases of Ebola virus in the population Acholi in Uganda already in 2003 [5].

These data stand for the demonstration that undoubtedly the rural populations have been in contact with EBOV for a long time so that they have had the possibility to establish towards this microorganism a physiological and immune response. This behavior is common in the zoonotic pathogenic agents which at the beginning lead to isolated cases of infection from animal to humans and later they evolve and spread from man to man on the basis of further genetic mutations.

Environmental and social variations as epidemic causes

Environmental characteristics that are similar for all zoonosis, have promoted the outbreak of the present EBOV infection. The reservoir of Zaire Ebolavirus has reported to be the bat of the fruit [6-9]. Although this question is still open, there are two possibilities for explaining the current epidemic: the bats in the forests around the prefecture of Gueckedou have been only recently infected or they have been already infected long since but the transmission to humans resulted to be more recent.

Following the most recent epidemiological and molecular data [10], it is possible to place the presence of Zaire EBOV in the forest near Gueckedou: the virus has spread from Central towards the North-West Africa during the last decade through one of the three bats of the fruit considered potential reservoir being able to migrate at long distance. Gire et al. [10] have demonstrated that some genomic heterogeneity

among various strains of Zaire EBOV can be detected and that the strains involved in the last three epidemiological sources of infection were different from those involved in previous epidemics nevertheless belonging to a common progenitor arisen in 2004. This evidence underlines the fact that all outbreaks of infections registered from 1976 up to now, derive from independent zoonotic events mainly due to new transmissions animal-man included in the same genetically heterogeneous source of virus present in the animal reservoir.

The queries that can be asked now are the following: 1) why unexpectedly did Zaire EBOV diffuse to West Africa whereas up to now it was exclusively observed in Central Africa? 2) Why did it appear in Guinea where it had not been observed? 3) Why only at this moment? The answers can rely once again on the surroundings in which the relationships between animal and human species occur.

The poverty of the populations in a certain area induces them to reach the forest but this situation increases the opportunities of contact between the individuals and the reservoirs of EBOV leading to a high risk of infection. Once infected, the humans can be a source of disease transmission for the rest of population which in turn is in a very unfavorable habitat due to the poor economic conditions. When the source of infection will be expanded enough to be evident at epidemiological level, the Government and the health facility due to poor economic conditions will be unable to stem the illness within and outside the country.

This event could be happened in Guinea at the beginning of the present epidemic when the first case of the child named Emile Ouamouno [2] has infected the doctor who was treating him in Meliandou village in the prefecture of Gueckedou. Afterwards Zaire EBOV began to spread to the neighboring countries such as Liberia and Sierra Leone both devastated by civil wars and Guinea that is considered one of the most indigent State in the world (United Nations Development Programme: http://undp.org/en/).

From the zoonosis to human Infectious Diseases

In order to consider the EBOV infection as a real emerging disease, the etiological agent may be fully adapted to human species and thus may be able to keep itself in a specific environment. Moreover it must replicate only in mankind having previously acquired the capacity to be efficiently transmitted from man to man. For doing this, two other steps will be necessary: first the pathogenic organism should be transmitted from the animal reservoir to humans and subsequently it should have the capacity to pass directly from man to man even if with a non-optimal efficiency. In the first step, the infected individual is defined dead-end host because the possibility of transmission is stopped and he is considered the final host of the pathogen (a classical example of this pathology is the rabies which affects canines and felines). At this stage, the microorganism may adapt itself to the new host acquiring the possibility to be transmitted directly from individual to individual causing small epidemic outbreaks. In recent years, the progression of similar situations has been seen in influenza H5N1 virus infection [11].The involved infectious disease stops to be diagnosed as a zoonosis and is considered as a human emerging disease in the third stage when the pathogenic agent is fully adapted to human species and consequently it efficiently spreads through individuals leading to epidemics of huge proportions or to pandemics .

Each step of this passage is due to genomic mutations that surely are more frequent in viruses than in bacteria [10]. For this reason, the shift from zoonosis to human emerging disease occurs more easily in the viral than in the bacterial infections or for other eukaryotic pathogens. Moreover the genomic material has the ability to continuously change thereby being subjected to the environmental pressure so that the co-evolution between the pathogen and the humans can indefinitely move on.

Besides further viral mutations might have enhanced the affinity towards the humans resulting in the presence of the current strains able to be transmitted to men with high efficiency.

At the moment, the evolution of Zaire EBOV in Africa is still in progress. In parallel with the epidemic occurred in December 2013 in the prefecture of Gueckedou in Guinea still ongoing, another one is reported in August 24th, 2014 in the district of Boende in the Democratic Republic of Congo where 49 deaths out of 69 total cases have been observed with a lethality rate of 71.01% [12]. In this second epidemic, the etiological agent was always Zaire EBOV but it was genetically different from that which now is spreading in West Africa; besides both viral variants differ respect to all strains isolated in the continent so far [2,13,14].

The two variants of the viruses are called respectively **Makona** for the strain in West Africa due to the name of river in Sierra Leone near to the border with Liberia and **Lomela** for the strain involved in epidemic in Central Africa in 2014 due to the name of river Lomela in the Democratic Republic of Congo [15]. The mortality rate of the variant Makona is at present about 40% whereas the variant Lomela shows a mortality of 75%; both these rates result to be completely different from previous epidemics data in which the mortality rate was higher (83-90%) [16]. The Makona mortality results significantly lower than that occurring at the beginning of the epidemic (50-53%) probably due to better therapeutic strategy[17,18].

In any case the historical general course of the disease is very interesting and the decrease of mortality rate of EBOV over time could emphasize a reduction of lethality in spite of its higher ability to spread in the environment.

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